<u>Drinking Water Quality: Trichloroethene (TCE) Contaminant Levels</u>

Type of EPHT	Hazard, Exposure
Indicator	
Measures	Level of Contaminant in Finished Water
	 Yearly distribution of number of CWS by maximum TCE concentration (cut-points: 0-<1, 1-<2, 2-<5, 5+ μg/L TCE).
	 Yearly distribution of number of CWS by mean TCE concentration (cut-points: 0-<1, 1-<2, 2-<5, 5+ μg/L TCE).
	3. Average Concentration of TCE, by Year.
	Potential Population Exposure to Contaminants in Finished Water 4. Yearly distribution of number of people served by CWS by maximum TCE concentration (cut-points: 0-<1, 1-<2, 2-<5, 5+ µg/L TCE).
	5. Yearly distribution of number of people served by CWS by mean TCE concentration (cut-points: 0-<1, 1-<2, 2-<5, 5+ μg/L TCE).
Derivation of Measures	TCE measures will be developed from water system attribute and water quality data stored in state Safe Drinking Water Act (SDWA) databases such as the Safe Drinking Water Information System (SDWIS/State). Data will be cleaned and transformed to a standard format. Analytical results of drinking water samples (usually taken at entry points to the distribution system or representative sampling points after treatment) will be used in conjunction with information about each CWS (such as service population and latitude and longitude of representative location of the CWS service area) to generate the measures.
Units	TCE, µg/L
Geographic Scope	State and Community Water System
Geographic Scale	The finest detail will be the approximate point location of the community water distribution system represented by water withdrawal point, water distribution extents, principal county served, or principal city served.
Time Period	2000 – Most Recent Year Available
Time Scale	Calendar year
Rationale	Trichloroethene (TCE) and Public Health
	Trichloroethene (TCE) is a volatile halogenated short-chain hydrocarbon. TCE is used primarily as an industrial degreaser, solvent, and in the synthesis of other chemicals. In the past, it was used in dry cleaning, food processing, household cleaners, and as a general anesthetic. TCE is produced and used in high volumes in the U.S. and has been detected in urban and ambient air and occasionally soils and drinking water most likely contaminated by industrial discharge (Moran et al., 2007; Rowe et al., 2007). Because of its volatility, this solvent does not persist in the soil or water following the discontinuation of contamination.
	Drinking or breathing high levels of TCE may cause nervous system effects, liver and lung damage, abnormal heartbeat, coma, and possibly death (ATSDR, 2003). Inhalation is the most common exposure route for the general population including

indoor sources from paints, adhesives, and cleaning solutions. Volatilization from contaminated water (e.g., shower water) as well as the use of household products containing this solvent can result in higher indoor than outdoor air concentrations (ATSDR, 1997b; Martin et al., 2005). Nearby dry cleaning establishments, industries producing this solvent, and contaminated waste disposal sites can also contribute to human exposure (Armstrong and Green, 2004; ATSDR, 1997a, 1997b, and 2000; Schreiber et al., 1993; Wallace et al., 1991). Drinking water may contribute to exposure when underground drinking water supplies have been contaminated. Workers in industries such as dry cleaning, aircraft maintenance, electronics manufacturing, and chemical production may be exposed by inhalation or dermal contact. The EPA has established drinking water standards and other environmental standards for TCE, and the FDA regulates TCE as an indirect food additive. OSHA has established workplace standards, and ACGIH has recommended occupational guidelines and biological exposure indices for monitoring workers (ACGIH, 2007). Human health effects from TCE at low environmental doses or at biomonitored levels from low environmental exposures are unknown. TCE is well absorbed by ingestion and inhalation, and animal studies have demonstrated that liquid forms can be dermally absorbed. Following absorption, part of the solvent dose is excreted into expired air (ATSDR1997a; Monster, 1986). The retained solvent can undergo hepatic metabolism. TCE is metabolized to trichloroacetic acid and tricholoroethanol, which are eliminated in the urine. Accidental or intentional high dose acute exposure by ingestion or inhalation can result in loss of motor coordination, somnolence, and unconsciousness. Inhaling high doses of TCE may also produce cardiac arrhythmias attributed to enhanced sensitivity to catecholamines. Prolonged, low level exposure to TCE has been associated with altered renal enzyme excretion and liver enlargement (ATSDR, 1997a, b). Chronic occupational exposure to TCE may be associated with mild degrees of neurological impairments, including reaction times, verbal skills, cognitive ability and motor function (Armstrong and Green, 2004). In animal studies, TCE induced kidney and liver tumors; and caused lung and testicular tumors (IARC, 1995). A recent EPA toxicological review (EPA/635/R-09/011F) characterized TCE as carcinogenic in humans by all routes of exposure (EPA, 2011). For cancer, the inhalation unit risk is 2×10^{-2} per ppm [4 × 10-6 per µg/m3], based on human kidney cancer risks (Charbotel et al.; 2006) and adjusted, using human epidemiologic data, for potential risk for non-Hodgkin lymphoma (NHL) and liver cancer. The oral unit risk for cancer is 5 x 10-2 per mg/kg/day, resulting from physiologically based pharmacokinetic model-based route-to-route extrapolation of the inhalation unit risk based on the human kidney cancer risks (Charbotel et al. 2006) and adjusted, using human epidemiologic data, for potential risk for NHL and liver cancer. There is high confidence in these unit risks for cancer, as they are based on good quality human data, as well as being similar to unit risk estimates based on multiple rodent bioassays. Evidence is sufficient to conclude that TCE operates through a mutagenic mode of action for kidney tumors. Evidence is insufficient and TCE-specific quantitative data are lacking on early-life susceptibility. Additional information about TCE is available from ATSDR at: http://www.atsdr.cdc.gov/toxprofiles/index.asp.

In an analysis of occurrence data from the EPA 6 Year Review of National Primary Drinking Water Regulations, TCE was detected in 1,013 systems serving 29.5 million people (EPA, 2009). Concentrations of TCE were greater than the MCL in 195 systems serving close to 12 million people. TCE was the fifth highest occurring regulated volatile organic chemical found based on the percent of population served by systems with at least one sample detection found from the 6 Year Review data (EPA, 2009).

Biomonitoring Information

Levels of halogenated solvents in blood reflect recent exposure. Blood levels of TCE

were generally not detected in the NHANES 2003-2004 subsample and were detected infrequently in previous U.S. surveys (CDC, 2009).

Comparatively higher blood levels of tetrachloroethene and TCE have been noted for urban and industrial residential settings than for rural settings (Barkley et al., 1980; Begerow et al., 1996; Brugnone et al., 1994). Finding a measurable amount of any of these solvents in blood does not mean that the level of the solvent causes an adverse health effect. Biomonitoring studies of blood halogenated solvents can provide physicians and public health officials with reference values so that they can determine whether people have been exposed to higher levels of halogenated solvents than levels found in the general population. Biomonitoring data can also help scientists plan and conduct research on exposure and health effects.

Sources of TCE

TCE does not occur naturally in the environment. However, it has been found in underground water sources and many surface waters as a result of the manufacture, use, and disposal of the chemical (ATSDR, 2003).

TCE Regulation and Monitoring

The EPA has set a maximum contaminant level for TCE in drinking water of 0.005 milligrams per liter (0.005 mg/L) or 5 parts of TCE per billion parts water. The EPA has also developed regulations for the handling and disposal of trichloroethylene.

OSHA has set an exposure limit of 100 parts of TCE per million parts of air (100 ppm) for an 8-hour workday, 40-hour work week (ATSDR, 2003).

Use of Measure

These measures can assist by addressing the following surveillance functions:

- Distribution measures provide information on the number of CWS and the number of people potentially exposed to TCE at different concentrations.
- Maximum concentrations provide information on the peak potential exposure to TCE at the state level.
- Mean concentrations at the CWS level provide information on potential exposure at a smaller geographic scale.

Limitations of The Measure

The current measures are derived for CWS only. Private wells may be another source of population exposure to TCE. Transient non-community water systems, which are regulated by EPA, also may be an important source of TCE exposure. Measures do not account for the variability in sampling, numbers of sampling repeats, and variability within systems. Concentrations in drinking water cannot be directly converted to exposure because water consumption varies by climate, level of physical activity, and between people (EPA 2004). Due to errors in estimating populations, the measures may overestimate or underestimate the number of affected people.

Data Sources

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Limitations of Data Sources

Ground water systems may have multiple wells with different TCE concentrations that serve different parts of the population. Compliance samples are taken at each entry point to the distribution system. In systems with separate wells serving some branches or sections of the distribution system, the system mean would tend to underestimate the TCE concentration of people served by wells with higher TCE concentrations. Exposure may be higher or lower than estimated if data from multiple entry points for water with different TCE levels are averaged to estimate levels for the PWS.

Related Indicators	Public Water Use
	ACGIH. TLVs and BEIs Based on the documentation of the threshold limit values for chemical substances and physical agents and biological exposure indices. 2007. Signature Publications. Cincinnati OH. p.104.
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	 Agency for Toxic Substances and Disease Registry (ATSDR). Toxicological profile for trichloroethylene update. 1997b [online]. Available at URL: http://www.atsdr.cdc.gov/toxprofiles/tp.asp?id=173&tid=30. 4/22/09
	 Agency for Toxic Substances and Disease Registry (ATSDR). ToxFAQs[™] for Trichloroethylene (TCE), July 2003. Available at: http://www.atsdr.cdc.gov/toxfaqs/tf.asp?id=172&tid=30
	 Agency for Toxic Substances and Disease Registry (ATSDR). Toxicological profile for Methylene chloride update. 2000 [online]. Available at URL: http://www.atsdr.cdc.gov/toxprofiles/tp.asp?id=234&tid=42. 4/22/09
	6. Armstrong SR, Green LC. Chlorinated hydrocarbon solvents. Clin Occup Environ Med 2004;4(3):481-496.
	 Barkley J, Bunch J, Bursey JT, Castillo N, Cooper SD, Davis JM, et al. Gas chromatography mass spectrometry computer analysis of volatilie halogenated hydrocarbons in man and his environment—a multimedia environmental study. Biomed Mass Spectrom 1980;7(4):139-147.
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	12. International Agency for Research in Cancer (IARC). IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, Dry Cleaning, Some Chlorinated Solvents and Other Industrial Chemicals Vol. 63, 1995. Available at: http://monographs.iarc.fr/ENG/Monographs/vol63/mono63.pdf
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	 Monster AC. Biological monitoring of chlorinated hydrocarbon solvents. J Occup Med 1986;28:583-588.
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	17. National Toxicology Program (NTP). Report on Carcinogens, 12th ed. 2010. [online]. Available at URL:
	http://ntp.niehs.nih.gov/ntp/roc/twelfth/ListedSubstancesReasonablyAnticipated

- 18. Rowe BL, Toccalino PL, Moran MJ, Zogorski JS, Price CV. Occurrence and potential human-health relevance of volatile organic compounds in drinking water from domestic wells in the United States. Environ Health Perspect 2007;115(11):1539-1546.
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